Treatment of accidental hypothermia with peritoneal dialysis

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When the rectal temperature falls below 35°C hypothermia is said to be present.[1] Accidental hypothermia is defined as an unintentional lowering of the body temperature due to exposure in a previously conscious patient.[2]

Because of the high mortality in profound accidental hypothermia[2] proper management of patients is crucial. However, the best method of treatment is controversial. Some authors have recommended active external rewarming of the patient by submersion in a warm bath[3] or the use of a hyperthermic mattress.[4] Others believe that slow passive external rewarming is adequate and recommend only that the patient be wrapped in a blanket.[5] Still others think that internal or core rewarming is the preferred treatment.[6] Methods of core rewarming include partial cardiopulmonary bypass,[6] thoracotomy and lavage of the mediastinum with warm fluids,[7] hemodialysis[8] and peritoneal dialysis.[9]

We report our experience with a patient whose recovery from accidental hypothermia coincided with peritoneal dialysis therapy.

Case report

A 20-year-old man with juvenile diabetes was brought to hospital after he was found sleeping in his neighbour's yard. He had consumed a large amount of alcohol the night before and had started to walk the four blocks home from his friend's house. The temperature that night was about −8°C.

The patient was semicomatose, with slow, shallow breathing. His skin was cold and acrocyanotic and his rectal temperature was 28°C (room temperature at the time was 23°C). Blood pressure was 110/70 mm Hg and the apical heart beat was regular at 120/min. His pupils were slightly dilated and sluggishly reactive to light. His muscles were stiff and his deep tendon reflexes were decreased.

Blood glucose concentration was 750 mg/dL and serum electrolyte concentrations were normal. Acetone was detected neither in the blood nor in the urine. Arterial blood gas measurements corrected for the patient's current temperature[10] were as follows: pH, 7.35; P_{CO_2}, 25 mm Hg; P_{O_2}, 74 mm Hg; and serum bicarbonate concentration, 13 mmol/L. Blood alcohol concentration was 300 mg/dL. An electrocardiogram showed sinus tachycardia and diffuse muscle tremors. A chest roentgenogram was normal.

The patient was wrapped in thick blankets but no active external means of rewarming the body was applied. Twenty minutes after the patient's admission his rectal temperature was still 28°C but his blood pressure had decreased to 90/60 mm Hg. Peritoneal dialysis was begun, with exchanges of 2 L of dialysate warmed to 38°C every 30 minutes. One hour after the initiation of dialysis the patient became alert and his rectal temperature increased to 33°C. Two hours later the temperature reached 37°C and dialysis was continued for another 5 hours. Thereafter the patient's temperature remained normal.

Discussion

Although our patient might have recovered simply because of re-entry into a warmer environment and the use of thick blankets, the prompt increase by 5°C in body temperature after an hour of peritoneal dialysis makes it likely that the dialysis was instrumental in the rapid and substantial improvement. With the use of blankets as a passive external means of rewarming it usually takes many more hours for the temperature to return to normal.[5]

We did not attempt to use active external methods of rewarming because current theory and experimental evidence appear to favour internal methods.[1,8,11] Rapid external rewarming tends to cause dilatation of constricted peripheral blood vessels, which can result in circulatory collapse, diversion of blood from the visceral organs,[8] and augmented return of cold venous blood from the skin and the extremities.[1] In persons rendered hypothermic by surface cooling, the temperature in the core is commonly higher than that in the periphery.[12] Consequently, early return of cold peripheral blood can result in further cooling of the core.[1] If the temperature of the heart is already close to lethal at the time of external rewarming, this further cooling might be sufficient to result in catastrophic arrhythmias or cardiac arrest.[1,11] Internal rewarming would probably avoid the above-mentioned complications.

Peritoneal dialysis, hemodialysis and partial cardiopulmonary bypass are all relatively simple methods of internal rewarming that are readily available in most hospitals. Hemodialysis and partial cardiopulmonary bypass could result in a faster return to normal of body temperature than peritoneal dialysis because of direct heating of blood. However, no studies comparing the efficacy of the three procedures have been carried out. In our patient the response to peritoneal dialysis was certainly prompt. Since hypotension is a common feature of accidental hypothermia[8] and since hemodialysis and cardiopulmonary bypass are fraught with problems and risks in the face of hypotension, peritoneal dialysis may prove to be the preferred treatment for patients with profound hypothermia and an unstable cardiovascular system. Indeed, in the only other report in the literature describing the use of peritoneal dialysis for rewarming, the blood pressure was undetectable at the beginning of dialysis.[9]

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References

1. BEESON PB, MCDERMOTT W (eds): *Textbook of Medicine*, 14th ed, Philadelphia, Saunders, 1975, pp 65-6
2. ZINGG W: The management of accidental hypothermia. *Can Med Assoc J* 96: 214, 1967
3. MERIWEATHER WD, GOODMAN RM: Severe accidental hypothermia with survival after rapid rewarming. Case report, pathophysiology and review of the literature. *Am J Med* 53: 505, 1972
4. FERNANDEZ JP, O'ROURKE RA, EWY GA: Rapid active external rewarming in accidental hypothermia. *JAMA* 212: 153, 1970
5. HUDSON LD, CONN RD: Accidental hypothermia — associated diagnoses and prognosis in a common problem. *JAMA* 227: 37, 1974
6. WICKSTROM P, RUIZ E, LILJA GP, et al: Accidental hypothermia — core rewarming with partial bypass. *Am J Surg* 131: 622, 1976
7. LINTON AL, LEDINGHAM IM: Severe hypothermia with barbiturate intoxication. *Lancet* 1: 24, 1966
8. THORN GW, ADAMS RD, BRAUNWALD E, et al (eds): *Harrison's Principles of Internal Medicine*, 8th ed, New York, McGraw, 1977, pp 57-8
9. LASH RF, BURDETTE JA, OZDIL T: Accidental profound hypothermia and barbiturate intoxication — a report of rapid "core" rewarming by peritoneal dialysis. *JAMA* 201: 123, 1967
10. SEVERINGHAUS JW: Blood gas concentrations, in *Handbook of Physiology*, section 3: *Respiration*, vol 2, FENN WO, RAHN H (eds), Washington, Am Physiol Soc, 1965, pp 1475-87
11. PATTON JF, DOOLITTLE WH: Core rewarming by peritoneal dialysis following induced hypothermia in the dog. *J Appl Physiol* 33: 800, 1972
12. HARDY JD, BARD P: Body temperature regulation, in *Medical Physiology*, 14th ed, vol 2, MOUNTCASTLE VB (ed), St Louis, Mosby, 1974, p 1312

Colchicine therapy for nephrotic syndrome due to familial Mediterranean fever

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Familial Mediterranean fever is a genetic disorder that occurs in certain ethnic groups (in descending order of frequency: Sephardic Jews, Armenians, Arabs, Ashkenazic Jews and others). It is marked by the sporadic appearance of acute attacks of fever and polyserositis and by the insidious onset of amyloidosis. Colchicine administration has been described as being effective in preventing acute attacks.[1-6] However, virtually no information exists regarding the effects of colchicine therapy on the associated amyloidosis.

In this report we describe a patient with familial Mediterranean fever in whom the nephrotic syndrome developed secondary to biopsy-proven renal amyloidosis. Improvement in both clinical features and proteinuria occurred with colchicine therapy.

Case report

A 42-year-old Italian woman presented for assessment of abdominal pain and peripheral edema. The pain had been present for approximately 20 years, occurring once or twice a month, lasting 4 to 5 days and usually being preceded by fever. The pain began in the epigastrium, rapidly spread throughout the abdomen and was associated with nausea and vomiting. Bilateral leg edema had been noticed by the patient for 2 months in association with facial puffiness and swelling of the hands.

Findings from systems review were otherwise unremarkable. Past medical history was negative except for appendectomy and hysterectomy at the ages of 15 and 22 years, respectively. The patient's brother had died at the age of 40 years of renal insufficiency of unknown origin.

Apart from moderate obesity and mild pretibial edema the results of physical examination were normal. Urinalysis revealed proteinuria (4+) and many hyaline and granular casts. The 24-hour urinary protein excretion was 16 and 18 g on two occasions. Serum concentrations were as follows: total protein, 4.8 g/dL; albumin, 1.6 g/dL; creatinine, 0.9 mg/dL; and cholesterol, 304 mg/dL. Additional extensive laboratory evaluation, including measurement of fasting and postprandial blood glucose concentrations and serum complement (C3 and C4) concentrations, and assessments for underlying collagen vascular disease, yielded normal results. Bilateral renal vein venography also yielded normal findings, thereby ruling out renal vein thrombosis as a cause of the nephrotic syndrome.

A renal biopsy specimen disclosed diffuse expansion of glomerular mesangial areas by homogeneous eosinophilic material that stained orange-red with Congo-red dye and exhibited the characteristic birefringence of amyloid (Fig. 1). Segments of glomerular basement membrane were thickened by the same material and the walls of small arteries contained amyloid. Electron microscopy revealed typical amyloid fibrils in the mesangium and in some segments of the glomerular basement membrane adjacent to the mesangium. Epithelial cell foot processes were effaced. Direct immunofluorescent examination of the specimen revealed diffuse deposits of C3 in the capillary walls and mesangium of glomeruli. There was no localization of immunoglobulins A, G or M or of fibrin. The findings were diagnostic of renal amyloidosis.

A diagnosis of familial Mediterranean fever with nephrotic syndrome secondary to renal amyloidosis was made and oral therapy with colchicine, 0.6 mg *tid*, was

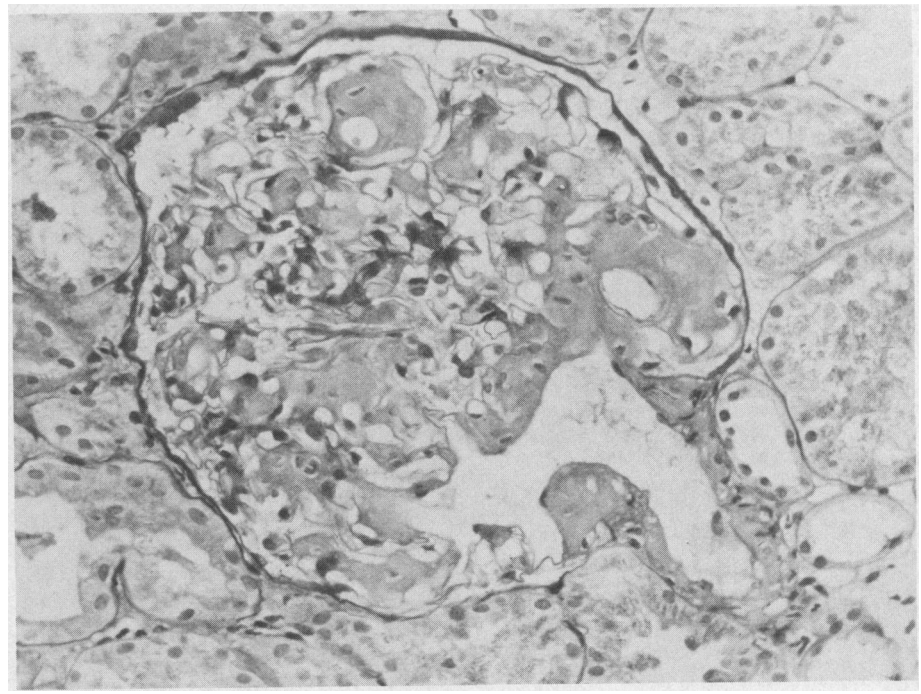

FIG. 1—Glomerular mesangium widened by amyloid deposits (periodic acid–Schiff; original magnification, ×160, reduced 20%).

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